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(FILE 'MEDLINE, HCAPLUS, BIOSIS, EMBASE, AGRICOLA, SCISEARCH' ENTERED AT  
12:26:17 ON 08 JUL 2003)

L22 29 DUP REM L21 (20 DUPLICATES REMOVED)

=> d que 122

L1 313 SEA VANDENBERGHE G?/AU  
L2 51 SEA VANDEN BERGHE G?/AU  
L3 692 SEA VAN DEN BERGHE G?/AU  
L4 1056 SEA (L1 OR L2 OR L3)  
L5 153 SEA L4 AND INSULIN#  
L6 123 SEA L5 AND CRITICAL?(3A) ILL?  
L7 1 SEA L6 AND NEUROPATH?  
L8 6 SEA CIPNP?  
L9 0 SEA L8 AND GLUCOSE#  
L10 132 SEA CRITICAL?(5A) ILL?(5A) NEUROPATH?  
L11 1 SEA L10 AND INSULIN?  
L12 1 SEA L10 AND GLUCOSE  
L13 473 SEA CRITICAL? AND ILL? AND NEUROPATH?  
L14 12 SEA L13 AND INSULIN?  
L15 9 SEA L13 AND GLUCOSE?  
L16 596 SEA ILL?(5A) NEUROPATH?  
L17 23 SEA L16 AND INSULIN?  
L18 9 SEA L16 AND GLUCOSE?  
L19 29483 SEA DIABET?(3A) NEUROPATH?  
L20 9 SEA L19 AND CRIT?(5A) ILL?  
L21 49 SEA L7 OR L9 OR L11 OR L12 OR L14 OR L15 OR L17 OR L18 OR L20  
L22 29 DUP REM L21 (20 DUPLICATES REMOVED)

=> d ibib abs 122 1-29

L22 ANSWER 1 OF 29 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2003226479 EMBASE

TITLE: Introduction.

AUTHOR: Le Roith D.

CORPORATE SOURCE: Dr. D. Le Roith, Clinical Endocrinology Branch, NIH,  
Bethesda, MD, United States

SOURCE: Clinical Cornerstone, (2003) 5/2 (vi-vii).

ISSN: 1098-3597 CODEN: CCLOAX

COUNTRY: United States

DOCUMENT TYPE: Journal; Editorial

FILE SEGMENT: 003 Endocrinology

017 Public Health, Social Medicine and Epidemiology

018 Cardiovascular Diseases and Cardiovascular Surgery

037 Drug Literature Index

038 Adverse Reactions Titles

LANGUAGE: English

L22 ANSWER 2 OF 29 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2002401295 EMBASE

TITLE: 48th Congress of the Slovak and Czech Society of Clinical  
Neurophysiology, Nove Zamky, 25-27 October 2001.

AUTHOR: Nevsimalova S.

CORPORATE SOURCE: S. Nevsimalova, First Medical Faculty, Department of  
Neurology, Katerinska 30, 120 00 Prague, Czech Republic.  
snevs@lf1.cuni.cz

SOURCE: Clinical Neurophysiology, (2002) 113/10 (1658-1663).

ISSN: 1388-2457 CODEN: CNEUFU

PUBLISHER IDENT.: S 1388-2457(02)00016-0

COUNTRY: Ireland  
DOCUMENT TYPE: Journal; Conference Article  
FILE SEGMENT: 008 Neurology and Neurosurgery  
030 Pharmacology  
032 Psychiatry  
037 Drug Literature Index  
LANGUAGE: English

L22 ANSWER 3 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE

1

ACCESSION NUMBER: 2003:168974 BIOSIS  
DOCUMENT NUMBER: PREV200300168974  
TITLE: Relationship of personality traits according to Tokyo University Egogram (TEG) to blood **glucose** control after the education of diabetic inpatient program in patients with type 2 diabetes mellitus.  
AUTHOR(S): Matsubayashi, Sunao (1); Mukuta, Toshio (1); Sakanaka, Akihito (1); Miyagawa, Shin-ichi (1); Kawai, Masayo (1)  
CORPORATE SOURCE: (1) Department of Psychosomatic Medicine, Fukuoka Tokushukai Medical Center, Fukuoka, Japan Japan  
SOURCE: Journal of the Japan Diabetes Society, (2002) Vol. 45, No. 11, pp. 783-789. print.  
ISSN: 0021-437X.  
DOCUMENT TYPE: Article  
LANGUAGE: Japanese

AB We evaluated the relationship of the personality trait according to Tokyo University Egogram. (TEG) to HbA1c in 61 type 2 diabetic patients of 42 men and 19 women (52.8+-12.0 years of age, 9.9+-9.5 years of **illness**, and 24.5+-4.5 kg/m2 of BMI) maximal up to 36 months after the education of diabetic inpatient. TEG is consisted of five categories of **critical** parent (CP), nurturing parent (NP), adult (A), free child (FC), and adapted child (AC). The complications were 25 patients for obese, 27 patients for **neuropathy**, 15 patients for retinopathy, 8 patients for nephropathy, and 17 patients for macro-angiopathy. The diet therapy alone, oral hypoglycemic agent, and **insulin** therapy were applied for 4 patients, 33 patients, and 24 patients, respectively. HbA1c from all patients decreased to 7.5+-1.8% at 3 months after the education from 10.5+-2.3% before the education. However, HbA1c gradually increased since 6 months after the education. Although there was no significant difference between two groups of the high score and the low score of CP, NP, A, or FC divided from the median, HbA1c from the group of high score of AC was significantly increased since 15 months after the education compared to that from the group of low score of AC. In conclusion, diabetic patients having the high score of AC related to the personality of low self esteem and/or co-dependant to others seems to be that their blood **glucose** control was gradually worsen although which initially improved due to the education applied to them.

L22 ANSWER 4 OF 29 SCISEARCH COPYRIGHT 2003 THOMSON ISI

ACCESSION NUMBER: 2002:828156 SCISEARCH  
THE GENUINE ARTICLE: 600ML  
TITLE: Nerve conduction studies in selected peripheral nerve disorders  
AUTHOR: Krarup C (Reprint)  
CORPORATE SOURCE: Rigshosp, Dept Clin Neurophysiol NF3063, Ctr Neurosci, Blegdamsvej 9, DK-2100 Copenhagen, Denmark (Reprint); Rigshosp, Dept Clin Neurophysiol NF3063, Ctr Neurosci, DK-2100 Copenhagen, Denmark  
COUNTRY OF AUTHOR: Denmark  
SOURCE: CURRENT OPINION IN NEUROLOGY, (OCT 2002) Vol. 15, No. 5, pp. 579-593.

Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST,  
PHILADELPHIA, PA 19106-3621 USA.  
ISSN: 1350-7540.

DOCUMENT TYPE: General Review; Journal  
LANGUAGE: English  
REFERENCE COUNT: 165

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB Purpose of review

The physiological properties of nerve and muscle are influenced by pathological changes and the aim of this review is to discuss recent contributions of electrophysiological studies to the understanding and diagnosis of selected peripheral nerve disorders. The relationships between pathology and physiology emphasize the close interdependence between electrophysiological studies, clinical deficits and other laboratory information. Attention should be paid to the strengths and limitations of electrophysiological methods, considering their impact on diagnosis and treatment of patients.

Recent findings

Several studies have shown particular pathophysiological profiles associated with different antibody subtypes in autoimmune peripheral neuropathies and this association further supports the suggestion of pathological specificity in both acute and chronic neuropathy. The sensitivity and specificity of physiological profiles therefore become increasingly important since some of these neuropathies are accessible to treatment. On the other hand, the pathophysiological and clinical profiles may be heterogeneous in patients with some disorders. This could be related to a more indistinct division between different types of pathology with increased understanding of pathogenetic mechanisms. Moreover, new insights into disturbed axonal function have stimulated attempts to develop methods to explore normal and diseased human nerve function in vivo.

Summary

The exploration of axonal membrane and ion-channel function has become accessible using studies of excitability and are of potential value where conventional studies only provide nonspecific evidence of the number of fibers and the integrity of myelin. These studies will presumably become increasingly important in the years ahead considering the lack of understanding of the functional disturbances in axonal neuropathies.

L22 ANSWER 5 OF 29 MEDLINE DUPLICATE 2  
ACCESSION NUMBER: 2002400727 MEDLINE  
DOCUMENT NUMBER: 22146941 PubMed ID: 12151692  
TITLE: Resolution of diabetic autonomic neuropathy.  
AUTHOR: Burden Mary L; Burden A C  
CORPORATE SOURCE: Birmingham Teaching Primary Care Trust, Heart of Birmingham  
PCG, 177 Church Hill Road, Handsworth, Birmingham B20 3PX,  
UK.. maryb@btinternet.com  
SOURCE: POSTGRADUATE MEDICAL JOURNAL, (2002 Jun) 78 (920) 360-1.  
Journal code: 0234135. ISSN: 0032-5473.  
PUB. COUNTRY: England: United Kingdom  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200209  
ENTRY DATE: Entered STN: 20020802  
Last Updated on STN: 20020912  
Entered Medline: 20020911

AB Many consider diabetic autonomic neuropathy to be an irreversible complication of diabetes of long duration. Three patients developed symptoms of autonomic neuropathy which subsequently resolved. Their autonomic neuropathy was not associated with long duration of diabetes,

but with weight loss. Each had marked weight loss and resolution occurred on regaining remembered premorbid weight. A woman aged 20 was admitted with anorexia nervosa (weight loss 6 kg). She complained of feeling bloated. Gastroenterological investigations showed delayed gastric emptying. RR ratio (respiration and standing) was abnormal. Resolution occurred after two years. A male aged 18 developed diabetic symptoms, which were overlooked. Twelve months later he presented underweight and ketonuric; **insulin** treatment was started but within one month he became impotent. Resolution occurred after 18 months. An 80 year old man presented after six months trial of diet and sulphonylurea therapy. He was underweight, had ketonuria, and such muscle loss that he was unable to sit unaided. **Insulin** treatment was started. He developed severe symptomatic postural hypotension. This resolved six months later by which time he had regained his normal weight. These cases **illustrated** symptomatic autonomic **neuropathy** occurring in relation to weight loss with resolution on recovery of normal weight, a temporal pattern mimicking that of acute cachectic painful neuropathy. Treatment of autonomic neuropathy should be like that of cachectic neuropathy, that is with an expectation of recovery and should include strategies to regain premorbid weight and achieve glycaemic control.

L22 ANSWER 6 OF 29 SCISEARCH COPYRIGHT 2003 THOMSON ISI  
 ACCESSION NUMBER: 2002:529170 SCISEARCH  
 THE GENUINE ARTICLE: 563GV  
 TITLE: A fuzzy-based methodology for the analysis of diabetic neuropathy  
 AUTHOR: Di Lascio L; Gisolfi A (Reprint); Albunia A; Galardi G; Meschi F  
 CORPORATE SOURCE: Univ Salerno, Dipartimento Matemat & Informat, Via Salvador Allende 64, I-84081 Baronissi, SA, Italy (Reprint); Univ Salerno, Dipartimento Matemat & Informat, I-84081 Baronissi, SA, Italy; Ist Sci San Raffaele, Dept Neurophysiol, I-20132 Milan, Italy; Ist Sci San Raffaele, Dept Pediat, I-20132 Milan, Italy  
 COUNTRY OF AUTHOR: Italy  
 SOURCE: FUZZY SETS AND SYSTEMS, (16 JUL 2002) Vol. 129, No. 2, pp. 203-228.  
 Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.  
 ISSN: 0165-0114.  
 DOCUMENT TYPE: Article; Journal  
 LANGUAGE: English  
 REFERENCE COUNT: 27

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB A new model for the fuzzy-based analysis of diabetic **neuropathy** is **illustrated**, whose pathogenesis so far is not well known. The underlying algebraic structure is a commutative 1-monoid, whose support is a set of classifications based on the concept of linguistic variable introduced by Zadeh. The analysis is carried out by means of patient's anagraphical and clinical data, e.g. age, sex, duration of the disease, **insulinic** needs, severity of diabetes, possible presence of complications. The results obtained by us are identical with medical diagnoses. Moreover, analyzing suitable relevance factors one gets reasonable information about the etiology of the disease, our results agree with most credited clinical hypotheses. (C) 2002 Elsevier Science B.V. All rights reserved.

L22 ANSWER 7 OF 29 MEDLINE DUPLICATE 3  
 ACCESSION NUMBER: 2002367628 MEDLINE  
 DOCUMENT NUMBER: 22108856 PubMed ID: 12116178  
 TITLE: Wolfram syndrome: identification of a phenotypic and

genotypic variant from Jordan.  
 AUTHOR: Ajlouni Kamel; Jarrah Nadim; El-Khateeb Mohammed; El-Zaheri Mohamed; El Shanti Hatem; Lidral Andrew  
 CORPORATE SOURCE: National Center for Diabetes, Endocrinology and Genetics, University of Jordan, Amman.. ajlouni@ju.edu.jo  
 SOURCE: AMERICAN JOURNAL OF MEDICAL GENETICS, (2002 May 30) 115 (1) 61-5.  
 Journal code: 7708900. ISSN: 0148-7299.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200301  
 ENTRY DATE: Entered STN: 20020713  
 Last Updated on STN: 20030129  
 Entered Medline: 20030128

AB Wolfram syndrome is an autosomal recessive disorder with probable locus heterogeneity. Only **insulin**-dependent diabetes mellitus and progressive optic-nerve atrophy are necessary to make the diagnosis, but associated findings include diabetes insipidus, sensorineural hearing loss, ataxia, peripheral **neuropathy**, urinary-tract atony, and psychiatric **illnesses**. We performed clinical and molecular studies on four consanguineous families with 16 affected individuals. We point out a new phenotypic variant with absent diabetes insipidus, presence of peptic ulcer disease and bleeding tendency secondary to a platelet aggregation defect. The same phenotypic variant turned out to be a genotypic variant with linkage to a second Wolfram syndrome locus (WFS2) on chromosome 4q22-24.  
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L22 ANSWER 8 OF 29 HCAPLUS COPYRIGHT 2003 ACS  
 ACCESSION NUMBER: 2001:833142 HCAPLUS  
 DOCUMENT NUMBER: 135:353239  
 TITLE: **Critical illness**  
**neuropathy** treatment with blood  
**glucose** regulators  
 INVENTOR(S): **Van Den Berghe, Greta**  
 PATENT ASSIGNEE(S): Novo Nordisk A/S, Den.; K.U. Leuven R + D  
 SOURCE: PCT Int. Appl., 41 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001085256	A2	20011115	WO 2001-DK287	20010430
WO 2001085256	A3	20020221		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
AU 2001054621	A5	20011120	AU 2001-54621	20010430
EP 1292324	A2	20030319	EP 2001-927641	20010430
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,			

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR  
 US 2002107178 A1 20020808 US 2001-853193 20010511  
 PRIORITY APPLN. INFO.: GB 2000-10856 A 20000505  
 DK 2001-604 A 20010415  
 DK 2001-605 A 20010416  
 WO 2001-DK287 W 20010430

AB This invention relates to a life saving medicament for **critically ill** patients and a method of treatment. The compn. is a pharmaceutically effective amt. of a blood **glucose** regulator which is used to control the blood **glucose** level. An examples is given of a clin. study in which the hypothesis that the incidence of **crit. illness neuropathy** can be reduced by more strict metab. using intensive **insulin** treatment from admission onward.

L22 ANSWER 9 OF 29 MEDLINE DUPLICATE 4  
 ACCESSION NUMBER: 2001256250 MEDLINE  
 DOCUMENT NUMBER: 21253057 PubMed ID: 11353882  
 TITLE: An unusual neuropathy in a diabetic patient: evidence for intravenous immunoglobulin-induced effective therapy.  
 AUTHOR: Romedenne P; Mukendi R; Stasse P; Indekeu P; Buysschaert M; Colin I M  
 CORPORATE SOURCE: Department of Internal Medicine, CHR-St Joseph Medical Center, Mons, Belgium.  
 SOURCE: DIABETES AND METABOLISM, (2001 Apr) 27 (2 Pt 1) 155-8. Journal code: 9607599. ISSN: 1262-3636.  
 PUB. COUNTRY: France  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200107  
 ENTRY DATE: Entered STN: 20010723  
 Last Updated on STN: 20010723  
 Entered Medline: 20010719

AB We report the case of a 68-year old type-2 diabetic male patient who was admitted to hospital for progressive weakness in the right lower limb. Although his metabolic control was good, he lost more than 20 kg of weight. Despite intensive physio- and vitaminotherapy, his neurological condition kept on degrading with a severe amyotrophy and pain of the right thigh. He was unable to walk and to stand alone. Besides a yet known sensitive polyneuropathy, the electrophysiological study revealed an obvious motor involvement with signs of demyelination and axonal degeneration. Combined with the albuminocytologic dissociation observed in the cerebrospinal fluid, these specific clinical and electrophysiological features led us to postulate a diagnosis of inflammatory neuropathy. The patient underwent a treatment by methylprednisolone and immunoglobins that rapidly induced a striking improvement of his neurological condition. This case report **illustrates** that rare forms of **neuropathy** such as inflammatory neuropathies close to chronic inflammatory demyelinating polyneuropathy (CIDP) can occur in diabetic patients and superimpose on the more commonly described forms of neuropathies. It recalls the importance of recognizing CIDP-like neuropathies because unlike other forms of neuropathy, inflammatory neuropathies are perfectly curable.

L22 ANSWER 10 OF 29 MEDLINE DUPLICATE 5  
 ACCESSION NUMBER: 2000312019 MEDLINE  
 DOCUMENT NUMBER: 20312019 PubMed ID: 10853552  
 TITLE: [Metabolic and nutritional neuropathies]. Neuropathies metaboliques et carentielles.  
 AUTHOR: Lagueny A

CORPORATE SOURCE: Service de neurologie Hopital du Haut-Leveque, Pessac..  
 alain.lagueny@chu-aquitaine.fr  
 SOURCE: REVUE DU PRATICIEN, (2000 Apr 1) 50 (7) 731-5.  
 Journal code: 0404334. ISSN: 0035-2640.  
 PUB. COUNTRY: France  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: French  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200007  
 ENTRY DATE: Entered STN: 20000720  
 Last Updated on STN: 20000720  
 Entered Medline: 20000711

AB The two main causes of metabolic **neuropathies** are successively **diabetes** and chronic renal insufficiency. **Diabetic neuropathies** include both diffuse polyneuropathies and focal neuropathies. Sensori(motor) polyneuropathy is the most frequent form and different therapeutic trials have been initiated on the ground of the vascular and metabolic factors implicated in its pathogenesis. Autonomic neuropathy is the major cause of morbidity and mortality. In patients with chronic renal failure, the polyneuropathy is improved by renal transplantation. The carpal tunnel syndrome is frequent in hemodialysis patients, and surgery gives the opportunity to look for beta-2-microglobulin amyloid deposits. Among the less frequent causes of peripheral neuropathies in which metabolic factors have been considered, we review hypoglycemia, chronic respiratory insufficiency due to chronic obstructive pulmonary disease, chronic liver diseases, and the polyneuropathy occurring in the **critically ill** patients with nutritional or metabolic failures. In chronic excessive drinkers peripheral neuropathy is classically associated with thiamine deficiency, but the direct effect of alcohol itself has been discussed. Various vitaminic deficiencies have been responsible for the development of peripheral neuropathies. The clinical forms often associate peripheral neuropathy with myelopathy, and serum vitamin E concentrations should be measured in patients with spinocerebellar disorders. Usually nutritional deficiencies need multivitamins supplementation.

L22 ANSWER 11 OF 29 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.  
 ACCESSION NUMBER: 2000409001 EMBASE  
 TITLE: Brain stem evoked responses in patients with diabetes mellitus.  
 AUTHOR: Sharma R.; Gupta S.C.; Tyagi I.; Kumar S.; Mukherjee K.  
 CORPORATE SOURCE: Dr. R. Sharma, Department of Neurosurgery, Sanjay Gandhi Post Grad. Institute, Lucknow 226 014, India  
 SOURCE: Indian Journal of Otolaryngology and Head and Neck Surgery, (2000) 52/3 (223-229).  
 Refs: 15  
 ISSN: 0019-5421 CODEN: IONSF6  
 COUNTRY: India  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 003 Endocrinology  
 005 General Pathology and Pathological Anatomy  
 008 Neurology and Neurosurgery  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English

AB Diabetes mellitus is a systemic disorder which targets multiple organs. The neurological involvement is not only in the form of peripheral neuropathy, but may also lead to central neuropathy. To evaluate the presence of central neuropathy auditory brain stem evoked responses were recorded from 25 normal hearing diabetic subjects aged 28 years to 49 years (Mean age 44.28 years) at 2KHz, 4KHz, and 6KHz, frequencies. The results obtained were compared with those obtained from 10 subjects with

normal hearing of matched age and sex. In diabetic subjects, abnormal wave latencies were correlated with blood **glucose** level, duration of **illness** and peripheral **neuropathy**. The absolute latencies and inter peak latencies were significantly impaired ( $P<.001$ ) in diabetic subjects as compared to control subjects at 2, 4 and 6KHz frequencies. The incidence of delayed wave latencies was 64%, 72% and 84% at 2KHz, 4KHz, and 6KHz respectively suggesting that if brain stem evoked response audiometry is conducted at higher frequency like 6KHz in diabetic patients, the involvement of central neural axis can be detected earlier. This study is the first to demonstrate that brain stem evoked response audiometry is a useful non-invasive test for earlier detection of damage in central neural axis in patients of diabetes mellitus. There is no relationship between the delayed wave latencies and the blood **glucose** level, however there exists a significant relationship between the delayed wave latencies and the duration of disease and peripheral neuropathy.

L22 ANSWER 12 OF 29 MEDLINE DUPLICATE 6  
 ACCESSION NUMBER: 2000013341 MEDLINE  
 DOCUMENT NUMBER: 20013341 PubMed ID: 10546018  
 TITLE: Cardiac autonomic dysfunction in diabetic children.  
 COMMENT: Comment in: Diabetes Care. 2000 Jul;23(7):1044-5  
 AUTHOR: Massin M M; Derkenne B; Tallsund M; Rocour-Brumioul D; Ernould C; Lebrethon M C; Bourguignon J P  
 CORPORATE SOURCE: Division of Pediatric Cardiology, University of Liege, Belgium.. martial.massin@chrcitadelle.be  
 SOURCE: DIABETES CARE, (1999 Nov) 22 (11) 1845-50.  
 Journal code: 7805975. ISSN: 0149-5992.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: (CLINICAL TRIAL)  
 (CONTROLLED CLINICAL TRIAL)  
 Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199912  
 ENTRY DATE: Entered STN: 20000113  
 Last Updated on STN: 20010716  
 Entered Medline: 19991208

AB OBJECTIVE: Adults with type 1 diabetes may have abnormal alterations in heart rate variability (HRV) due to cardiac autonomic **neuropathy**. This prospective study was performed to determine whether HRV can be used to detect subclinical autonomic **neuropathy** in diabetic children. RESEARCH DESIGN AND METHODS: We examined five time domain and three frequency domain HRV indices determined from 24-h Holter recordings in 73 diabetic children and adolescents aged 3-18 years (mean 12.1 years) with a mean duration of diabetes of 55 months. The measures were compared with normal ranges. Z scores were established for each parameter and were compared with classic risk factors of other diabetic complications. RESULTS: Most HRV indices were significantly depressed in children aged  $> \text{or} = 11$  years, and the levels of HRV abnormalities were significantly correlated with long-term metabolic control (mean GHb for 4 years) in that age-group. In younger patients, HRV indices were within the normal range and were not correlated with the level of metabolic control. **Illness** duration and microalbuminuria but not short-term metabolic control (most recent GHb) were also independently predictive of HRV abnormalities. CONCLUSIONS: These results suggest that early puberty is a **critical** period for the development of diabetic cardiac autonomic dysfunction. Therefore, all type 1 diabetic patients should be screened for this complication by HRV analysis beginning at the first stage of puberty regardless of **illness** duration, microalbuminuria, and level of metabolic control.



L22 ANSWER 13 OF 29 MEDLINE  
 ACCESSION NUMBER: 2000126692 MEDLINE  
 DOCUMENT NUMBER: 20126692 PubMed ID: 10660855  
 TITLE: Reversible tetraplegia due to polyneuropathy in a diabetic patient with hyperosmolar non-ketotic coma.  
 AUTHOR: Kennedy D D; Fletcher S N; Ghosh I R; Coakley J H; Monson J P; Hinds C J  
 CORPORATE SOURCE: Department of Intensive Care, St. Bartholomew's Hospital, Smithfield, London, UK.  
 SOURCE: INTENSIVE CARE MEDICINE, (1999 Dec) 25 (12) 1437-9.  
 Journal code: 7704851. ISSN: 0342-4642.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200002  
 ENTRY DATE: Entered STN: 20000314  
 Last Updated on STN: 20000314  
 Entered Medline: 20000229

AB **Critical illness** polyneuromyopathy has not previously been reported as a complication of diabetic coma. We describe a patient with hyperosmolar non-ketotic coma (HONK) complicating gram-negative sepsis in whom persistent coma and profound tetraplegia caused considerable concern. Although, initially, it was feared that the patient had suffered a central neurological complication such as stroke or cerebral oedema, a diagnosis of **critical illness** motor syndrome (CIMS) was subsequently confirmed neurophysiologically. Profound limb weakness associated with HONK is not necessarily due to a catastrophic cerebral event, rather it may be a result of CIMS, which has an excellent prognosis for full neurological recovery.

L22 ANSWER 14 OF 29 SCISEARCH COPYRIGHT 2003 THOMSON ISI  
 ACCESSION NUMBER: 1999:803972 SCISEARCH  
 THE GENUINE ARTICLE: 246TJ  
 TITLE: Altered brain activation in cognitively intact individuals at high risk for Alzheimer's disease  
 AUTHOR: Smith C D (Reprint); Andersen A H; Kryscio R J; Schmitt F A; Kindy M S; Blonder L X; Avison M J  
 CORPORATE SOURCE: UNIV KENTUCKY, MED CTR, MRISC 113, 800 ROSE ST, LEXINGTON, KY 40536 (Reprint); UNIV KENTUCKY, COLL MED, DEPT NEUROL, LEXINGTON, KY; UNIV KENTUCKY, COLL MED, DEPT STAT, LEXINGTON, KY; UNIV KENTUCKY, COLL MED, DEPT BIOCHEM, LEXINGTON, KY; UNIV KENTUCKY, COLL MED, DEPT BEHAV SCI, LEXINGTON, KY; UNIV KENTUCKY, COLL MED, MAGNET RESONANCE IMAGING & SPECT CTR, LEXINGTON, KY; UNIV KENTUCKY, COLL MED, SANDERS BROWN CTR AGING, LEXINGTON, MA  
 COUNTRY OF AUTHOR: USA  
 SOURCE: NEUROLOGY, (22 OCT 1999) Vol. 53, No. 7, pp. 1391-1396.  
 Publisher: LIPPINCOTT WILLIAMS & WILKINS, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106.  
 ISSN: 0028-3878.  
 DOCUMENT TYPE: Article; Journal  
 FILE SEGMENT: LIFE; CLIN  
 LANGUAGE: English  
 REFERENCE COUNT: 32

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB Objective: To determine whether brain function is altered in cognitively normal individuals at high risk for AD several years before the typical age at onset for this **illness**. Background: **Neuropathologic** alterations in AD precede cognitive impairment by

several years. It is unknown whether functional alterations in neural circuitry accompany these neuropathologic changes, and if so, whether they may be detectable before onset of symptoms. Methods: We used functional MRI to compare cortical activation between two groups of cognitively normal women differing only in their risk for developing AD. Visual naming and letter fluency tasks were used to activate brain areas subserving object and face recognition, previously described sites of hypometabolism and neuropathologic alteration in AD. The risk groups differed in family history of AD and apolipoprotein E allele status, but were matched in age, education, and measures of cognitive performance. Average age of the study participants was 52 years. Results: The regional patterns of brain activation were similar between groups. However, the high risk group showed areas of significantly reduced activation in the mid- and posterior inferotemporal regions bilaterally during both tasks despite identical naming and letter fluency performance. Conclusions: Cognitively normal individuals at high risk for AD demonstrate decreased brain activation in key areas engaged during naming and fluency tasks. Decreased activation in the high risk group may be a consequence of the presence of subclinical neuropathology in the inferotemporal region or in the inputs to that region. If so, these findings provide evidence of a window of opportunity for disease-modifying treatment before the onset of symptomatic AD.

L22 ANSWER 15 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
7

ACCESSION NUMBER: 2000:131021 BIOSIS  
DOCUMENT NUMBER: PREV200000131021  
TITLE: Sympathetic skin response in diabetic children: Do diabetic children have diabetic neuropathy.  
AUTHOR(S): Torigoe, Katsumi (1); Numata, Osamu; Yazaki, Satoshi; Hasegawa, Satoshi; Boku, Naoki; Hiura, Makoto; Ino, Haruyoshi; Matsunaga, Masamichi  
CORPORATE SOURCE: (1) Department of Pediatrics, Nagaoka Red Cross Hospital, 297-1 Terajima-machi, Nagaoka, Niigata, 940-2085 Japan  
SOURCE: Pediatrics International., (Dec., 1999) Vol. 41, No. 6, pp. 631-636.  
ISSN: 1328-8067.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AB Background: Abnormal sympathetic skin response (SSR) has been reported in adult patients with diabetic neuropathy. In addition, other studies have revealed abnormal SSR in diabetic patients not having autonomic symptoms and autonomic dysfunctions. These findings have been only obtained from adult patients. There have been few reports on the autonomic functions in diabetic children. Accordingly, it is not clear whether the autonomic neuropathy occurs in diabetic children. The aim of the present study is to clear autonomic function in children with **insulin**-dependent diabetes mellitus by SSR. Methods: The SSR was measured in 28 normal healthy children and in eight patients with IDDM not having symptoms of dysautonomia. The SSR was elicited using 10 stimuli on programmed Nihonkoden Neuropack Sigma model machine. Following a single electrical stimulation, four SSR were recorded in both the palms and the soles simultaneously. Results: The SSR were simultaneously obtained in 100% of the two groups. The amplitudes in the palms and soles were not significantly different between the two groups. The mean and shortest latency in the soles were significantly longer in the IDDM group than in the control group ( $P < 0.01$ ). None of the measurements of SSR revealed correlation with duration of diabetes and onset of **illness**. Conclusions: Diabetic **neuropathy** may not have occurred in young patients having shorter duration of illness. Conversely, assuming that prolonged latency is abnormal, it may even have occurred in them. Follow

up on these patients with prolonged latencies would be required.

L22 ANSWER 16 OF 29 MEDLINE DUPLICATE 8  
ACCESSION NUMBER: 1999358665 MEDLINE  
DOCUMENT NUMBER: 99358665 PubMed ID: 10431718  
TITLE: **Critical** evaluation of the streptozotocin model  
of painful diabetic **neuropathy** in the rat.  
AUTHOR: Fox A; Eastwood C; Gentry C; Manning D; Urban L  
CORPORATE SOURCE: Novartis Institute for Medical Sciences, London, UK..  
alyson.fox@pharma.novartis.com  
SOURCE: PAIN, (1999 Jun) 81 (3) 307-16.  
Journal code: 7508686. ISSN: 0304-3959.  
PUB. COUNTRY: Netherlands  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199909  
ENTRY DATE: Entered STN: 19991005  
Last Updated on STN: 19991005  
Entered Medline: 19990923

AB Streptozotocin (STZ)-induced diabetes in the rat has been increasingly  
used as a model of painful diabetic **neuropathy** to assess the  
efficacies of potential analgesic agents. We have established this model,  
and here we question whether the changes in nocifensive reflex activity,  
used as a measure of hyperalgesia, are genuinely indicative of peripheral  
**neuropathy** or may rather be attributed to the extreme poor health  
of the animals. For comparison we have examined animals with peripheral  
**neuropathy** induced by partial ligation of the sciatic nerve.  
Diabetic animals were chronically ill, with reduced growth rate,  
polyuria, diarrhoea, and had enlarged and distended bladders. Indicative  
of their poor health, diabetic animals showed markedly reduced motor  
activity. In contrast, following partial sciatic nerve ligation rats  
showed none of these adverse effects and their motor activity was not  
different to naive animals. Diabetic animals displayed marked mechanical  
hyperalgesia, and some thermal hypoalgesia. Morphine and L-baclofen  
partially reversed established STZ-induced mechanical hyperalgesia, whilst  
the NK-1 receptor-antagonist RP-67580, the NMDA-antagonists MK801 and  
ketamine, and the nitric oxide synthase inhibitor L-NAME were without  
significant effect. Morphine and L-baclofen produced greater reversal of  
mechanical hyperalgesia following partial nerve ligation, although RP67580  
and MK801 showed little or no activity. These data confirm previous  
findings that STZ-induced diabetes in rats produces long-lasting  
mechanical, but not thermal hyperalgesia. In our experience this  
mechanical hyperalgesia is largely resistant to a range of pharmacological  
tools. However, we feel that the profound ill-health of the  
animals, together with the poor activity of a range of potential analgesic  
drugs, must raise questions on the predictive value of these animals as a  
model for the human condition of chronic diabetic pain seen in patients  
receiving long-term **insulin** treatment, as well as ethical  
concerns on the use of the animals themselves.

L22 ANSWER 17 OF 29 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.  
ACCESSION NUMBER: 1998207563 EMBASE  
TITLE: Long-term complications in newly diagnosed Sri Lankan  
patients with type 2 diabetes mellitus.  
AUTHOR: Weerasuriya N.; Siribaddana S.; Dissanayake A.; Subasinghe  
Z.; Wariyapola D.; Fernando D.J.S.  
CORPORATE SOURCE: Prof. D.J.S. Fernando, 53A Flower Road, Colombo 7, Sri  
Lanka. devakaf@eureka.lk  
SOURCE: QJM - Monthly Journal of the Association of Physicians,  
(1998) 91/6 (439-443).

Refs: 36  
 ISSN: 0033-5622 CODEN: QMJPFH  
 COUNTRY: United Kingdom  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 003 Endocrinology  
 006 Internal Medicine  
 017 Public Health, Social Medicine and Epidemiology  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English

AB We screened 597 newly-diagnosed diabetic patients (201 women) mean  $\pm$  SD age 42.3  $\pm$  6.2 years to determine the prevalence of diabetic complications; 22% presented because of symptoms of diabetes, 27% were diagnosed when hyperglycaemia was discovered at a health screening, and 36% were diagnosed while being treated for intercurrent illness. **Neuropathy** was present in 25.1%, nephropathy in 29%, retinopathy in 15%, coronary vascular disease in 21%, stroke in 5.6%, peripheral vascular disease in 4.8%, hypertension in 23%, obesity in 16%, central obesity in 21.3%, hypercholesterolaemia in 11%, hypertriglyceridaemia in 14%, and low high-density lipoprotein cholesterol in 12%. The prevalence of coronary vascular disease, hypertension, stroke, neuropathy and retinopathy at the time of diagnosis were higher in our patients than in Caucasian and Indo-Asian patients in the UK. Both a genetic predisposition to develop complications, and exposure to a longer duration of asymptomatic hyperglycaemia due to poor access to adequate health care, may contribute to the high frequency of complications at diagnosis. Since complications are already present at diagnosis, there is a case for implementing primary prevention programmes combined with screening for diabetes in high-risk groups.

L22 ANSWER 18 OF 29 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 1998058252 EMBASE  
 TITLE: Heart rate variability - A potential, noninvasive prognostic index in the **critically ill** patient.  
 AUTHOR: Kennedy H.L.  
 CORPORATE SOURCE: Dr. H.L. Kennedy, Cardiovascular Research Foundation, St. John's Mercy Medical Center, 777 So. New Ballas Road, St. Louis, MO 63141, United States  
 SOURCE: Critical Care Medicine, (1998) 26/2 (213-214).  
 Refs: 12  
 ISSN: 0090-3493 CODEN: CCMD7  
 COUNTRY: United States  
 DOCUMENT TYPE: Journal; Editorial  
 FILE SEGMENT: 005 General Pathology and Pathological Anatomy  
 008 Neurology and Neurosurgery  
 018 Cardiovascular Diseases and Cardiovascular Surgery  
 LANGUAGE: English

L22 ANSWER 19 OF 29 SCISEARCH COPYRIGHT 2003 THOMSON ISI

ACCESSION NUMBER: 96:597638 SCISEARCH  
 THE GENUINE ARTICLE: VB010  
 TITLE: THE WERNICKE-KORSAKOFF-SYNDROME  
 AUTHOR: BRODY B A (Reprint)  
 CORPORATE SOURCE: NORTHWESTERN UNIV, SCH MED, DEPT PATHOL, DIV NEUROPATHOL, CHICAGO, IL, 60611  
 COUNTRY OF AUTHOR: USA  
 SOURCE: INTERNATIONAL JOURNAL OF NEURORADIOLOGY, (MAY/JUN 1996)  
 Vol. 2, No. 3, pp. 216-230.  
 ISSN: 1079-8110.  
 DOCUMENT TYPE: General Review; Journal  
 LANGUAGE: ENGLISH

REFERENCE COUNT: 114

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB The Wernicke-Korsakoff syndrome is a complex neurologic disorder caused by nutritional thiamine deficiency producing a characteristic, bilaterally symmetrical distribution of lesions within the medial diencephalon, brain stem, and cerebellum. The **neuropathology** of three clinically undiagnosed cases is described to **illustrate** the range of clinical presentation and associated **neuropathology** seen in acute, acute superimposed on chronic, and subacute chronic disease. The anatomically selective pathogenesis of these lesions is based on energy failure from blocked **glucose** metabolism because of regionally specific depletion of thiamine-diphosphate and alpha-ketoglutarate dehydrogenase. The energy failure ultimately contributes to increased histamine release, glutamate accumulation, and neuronal damage via glutamate-mediated calcium channel excitotoxicity. The vascular damage that dominates the pathology of acute severe thiamine depletion is reversible. The more subtle parenchymal damage, once initiated, can progress onward to cause irreversible neurologic damage. It may also result from waxing and waning subacute levels of thiamine deficiency. In acute cases, the timing of treatment is **critical** in determining the extent of residual damage. Magnetic resonance imaging (MRI) may now help to achieve earlier clinical diagnosis.

L22 ANSWER 20 OF 29 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.DUPLICATE 9

ACCESSION NUMBER: 94366229 EMBASE

DOCUMENT NUMBER: 1994366229

TITLE: Lithotomy position-induced femoral neuropathy.

AUTHOR: Goh J.T.W.; Gregora M.G.; Welch M.

CORPORATE SOURCE: Royal Women's Hospital, Bowen Bridge Road, Herston, QLD 4029, Australia

SOURCE: Australian and New Zealand Journal of Obstetrics and Gynaecology, (1994) 34/5 (596-597).  
ISSN: 0004-8666 CODEN: AZOGBS

COUNTRY: Australia

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 009 Surgery  
010 Obstetrics and Gynecology  
033 Orthopedic Surgery

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Femoral neuropathy can occur following placement in the lithotomy position and has been rarely reported following vaginal hysterectomy. Contributing factors to this condition include positioning of the patient, duration of surgical procedure and **glucose** intolerance. The prognosis of lithotomy position induced femoral **neuropathy** is excellent. An **illustrative** case is herein reported.

L22 ANSWER 21 OF 29 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 94217293 EMBASE

DOCUMENT NUMBER: 1994217293

TITLE: Management of the trauma victim with pre-existing endocrine disease.

AUTHOR: Boulanger B.K.; Gann D.S.

CORPORATE SOURCE: Department of Surgery, Univ. of Maryland School of Medicine, 22 South Greene Street, Baltimore, MD 21201, United States

SOURCE: Critical Care Clinics, (1994) 10/3 (537-554).  
ISSN: 0749-0704 CODEN: CCCLEH

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 003 Endocrinology

024 Anesthesiology  
 030 Pharmacology  
 037 Drug Literature Index  
 049 Forensic Science Abstracts

LANGUAGE: English  
 SUMMARY LANGUAGE: English

AB The clinical course of individuals after trauma largely is determined by their pretraumatic state. The endocrine system plays a major role in the response to injury, surgery, and sepsis, and endocrine dysfunction places the trauma victim at risk of greater morbidity and mortality. Further, chronic endocrine disease usually is accompanied by multiorgan dysfunction, which may compromise the physiologic reserve of the **critically** injured patient. Among patients with pre-existing endocrine disease, the severe stresses of multisystem trauma can lead to a further, often subtle, decompensation in endocrine function. In the management of trauma victims with pre-existing endocrine disease, the role of the **critical** care specialist is three-fold- (1) to maintain a high index of suspicion for endocrine disease in all trauma victims, (2) to anticipate and prevent endocrine organ decompensation, and (3) to rapidly diagnose and institute therapy in those suspected of having endocrine disease.

L22 ANSWER 22 OF 29 SCISEARCH COPYRIGHT 2003 THOMSON ISI

ACCESSION NUMBER: 91:355800 SCISEARCH

THE GENUINE ARTICLE: FR874

TITLE: CARBOHYDRATE-RELATED POLYNEUROPATHY IN INTENSIVE-CARE PATIENTS

AUTHOR: WALDHAUSEN E (Reprint); KESER G

CORPORATE SOURCE: JOHANNA ETIENNE KRANKENHAUS, ANAESTHESIE ABT, AM HASENBERG 46, W-4040 NEUSS, GERMANY (Reprint)

COUNTRY OF AUTHOR: GERMANY

SOURCE: ANAESTHESIST, (1991) Vol. 40, No. 6, pp. 332-338.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: CLIN

LANGUAGE: German

REFERENCE COUNT: 72

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB In 30 septic, edematous intensive care patients a polyneuropathy occurred during treatment of peritonitis, pancreatitis, adult respiratory distress syndrome, or bronchopneumonia; 28 patients developed a complete tetraplegia. We believe this **neuropathy** to be an important cause of weaning failure. All patients had received parenteral or enteral nutrition with 240-800 g carbohydrate per day. Clinical data indicate that impairment of carbohydrate metabolism was the essential cause of the polyneuropathy. In 14 patients carbohydrate administration was continued; 13 died without neuromuscular recovery. In 16 patients carbohydrate nutrition was reduced to 100-250 g per day after the occurrence of tetraplegia; 13 of these made a full neurologic recovery.

L22 ANSWER 23 OF 29 SCISEARCH COPYRIGHT 2003 THOMSON ISI

ACCESSION NUMBER: 91:33287 SCISEARCH

THE GENUINE ARTICLE: EQ853

TITLE: PERIPHERAL-NERVE FUNCTION IN SEPSIS AND MULTIPLE ORGAN FAILURE

AUTHOR: WITT N J; ZOCHODNE D W; BOLTON C F (Reprint); MAISON F G; WELLS G; YOUNG G B; SIBBALD W J

CORPORATE SOURCE: HLTH & WELF CANADA, CTR DIS CONTROL LAB, OTTAWA K1A 0L2, ONTARIO, CANADA; UNIV WESTERN ONTARIO, DEPT CLIN NEUROL SCI, LONDON N6A 3K7, ONTARIO, CANADA; UNIV WESTERN ONTARIO, DEPT MED, LONDON N6A 3K7, ONTARIO, CANADA

COUNTRY OF AUTHOR: CANADA

SOURCE: CHEST, (1991) Vol. 99, No. 1, pp. 176-184.  
DOCUMENT TYPE: Article; Journal  
FILE SEGMENT: LIFE; CLIN  
LANGUAGE: ENGLISH  
REFERENCE COUNT: 51

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB Forty-three patients who had sepsis and multiple organ failure (**critical illness**) were studied prospectively to determine the incidence and severity of peripheral nerve function and to correlate such function with a number of variables. Electrophysiologic studies indicated a primary axonal degeneration of motor and sensory fibers in 30 (70 percent). Fifteen (30 percent) had the clinical signs of difficulty in weaning from assisted ventilation, weakness of limb muscles, and reduced or absent deep tendon reflexes. Full recovery from the polyneuropathy occurred among the 23 (53 percent) who survived, except three who had a very severe polyneuropathy. A peripheral nerve function index, computed from electrophysiologic measurements, showed statistically significant ( $p < 0.01$ ) negative correlations with the time in the **critical** care unit, and the serum **glucose** value; the serum albumin level showed a positive correlation. Multiple regression analyses indicated all three factors accounted for 47 percent ( $r^2 = 0.4678$ ) of all potential variables. In a separate analysis, the nerve function index correlated with the amplitude of the diaphragm compound muscle action potential ( $p < 0.01$ ). The results were consistent with the polyneuropathy being due to the same mechanisms that are currently postulated to cause dysfunction in this syndrome of other organ systems (including the neuromuscular respiratory system).

L22 ANSWER 24 OF 29 MEDLINE DUPLICATE 10  
ACCESSION NUMBER: 91005695 MEDLINE  
DOCUMENT NUMBER: 91005695 PubMed ID: 2209327  
TITLE: Relationship of psychiatric illness to impotence in men with diabetes.  
AUTHOR: Lustman P J; Clouse R E  
CORPORATE SOURCE: Department of Psychiatry, Washington University School of Medicine, St. Louis, Missouri.  
CONTRACT NUMBER: DK-36452 (NIDDK)  
RR-00036 (NCRR)  
SOURCE: DIABETES CARE, (1990 Aug) 13 (8) 893-5.  
Journal code: 7805975. ISSN: 0149-5992.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199011  
ENTRY DATE: Entered STN: 19910117  
Last Updated on STN: 19910117  
Entered Medline: 19901121

AB Clinical data from 37 adult males with diabetes mellitus (**insulin** dependent,  $n = 22$ ; non-**insulin** dependent,  $n = 15$ ) who had undergone psychiatric diagnosis and peripheral nerve conduction studies were reviewed to determine whether psychiatric illness was significantly related to complaints of sexual dysfunction. Main-effects testing revealed that impotence was associated with both neuropathy ( $P$  less than 0.01) and psychiatric illness ( $P$  less than 0.001). Logistic regression analysis was then used to determine the independent relationships of these two variables with impotence. After controlling for the effects of **neuropathy**, psychiatric **illness** (generalized anxiety disorder and depression) remained significantly associated with sexual dysfunction ( $P$  less than 0.01). These data allow for the hypothesis that psychiatric illness may be an important contributor to impotence in

diabetic men, as it is in nondiabetic men, even when neuropathic complications of the disease are present.

L22 ANSWER 25 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 1987:294290 BIOSIS  
DOCUMENT NUMBER: BA84:24322  
TITLE: SIADH IN A DIABETIC PATIENT WITH SEVERE NEUROPATHY.  
AUTHOR(S): INOUE K; WASADA T; NAKAGAWA M; TAZOE A; ONO H; INOUCHI T;  
UMEDA F; IBAYASHI H  
CORPORATE SOURCE: THIRD DEP. INTERNAL MED., FAC. MED., KYUSHU UNIVERSITY,  
FUKUOKA 812, JAPAN.  
SOURCE: J JPN DIABETES SOC, (1987) 30 (2), 181-185.  
CODEN: TONYA4. ISSN: 0021-437X.  
FILE SEGMENT: BA; OLD  
LANGUAGE: Japanese

AB A 55 year-old woman, who had had diabetes for 2 years, was admitted to our hospital because of marked emaciation (body mass index 13.6) and painful **neuropathy**. She was chronically ill and apathetic. With daily **insulin** treatment (14-16 U), her fasting blood **glucose** was 150 mg/dl and HbA1 9.6%. Renal and endocrine functions including the thyroid gland, the pituitary adrenocortical axis and the renin aldosterone system were normal. She had the clinical features of severe symmetric peripheral neuropathy. The MCV of posterior tibial nerve was 32.4 m/sec. The SCV of the sural nerve could not be evoked. The R-R interval variation during deep breathing was 2.3 beats/min. Biopsy of the sural nerve revealed a marked loss of both myelinated and unmyelinated nerve fibers, independent of fiber size. Her blood showed low Na concentrations (127-131 mEq/l) and hypo-osmolality (236-256 mOsm/l). Water loading (20 ml/kg) failed to dilute the urine below the level of the plasma osmolality and the plasma ADH was not significantly suppressed. Urinary Na output was relatively large (28-120 mEq/l) in the presence of hyponatremia. Interestingly, these abnormalities in the water loading test persisted for 2 months after discontinuation of treatment with carbamazepine, when serum Na had already been normalized. These findings suggest that a certain causal linkage may exist between inappropriate secretion of ADH and severe diabetic neuropathy.

L22 ANSWER 26 OF 29 MEDLINE  
ACCESSION NUMBER: 86165639 MEDLINE  
DOCUMENT NUMBER: 86165639 PubMed ID: 3956933  
TITLE: Correlation of esophageal motility abnormalities with neuropsychiatric status in diabetics.  
AUTHOR: Clouse R E; Lustman P J; Reidel W L  
CONTRACT NUMBER: AM07130 (NIADDK)  
AM20579 (NIADDK)  
AM31496 (NIADDK)  
+  
SOURCE: GASTROENTEROLOGY, (1986 May) 90 (5 Pt 1) 1146-54.  
Journal code: 0374630. ISSN: 0016-5085.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 198605  
ENTRY DATE: Entered STN: 19900321  
Last Updated on STN: 19970203  
Entered Medline: 19860515

AB Esophageal motility abnormalities, **neuropathy**, and psychiatric **illness** were independently determined in 30 patients with type 1 or type 2 diabetes mellitus to clarify the interrelationship of these findings in diabetics. Fifteen patients (50%) were found to have



esophageal contraction abnormalities, a specific cluster of manometric derangements. Diagnoses of depression, dysthymia, or generalized anxiety disorder were made in 87% of those with contraction abnormalities but in only 21% of the patients with normal manometric patterns ( $p = 0.002$ ). Log-linear analysis confirmed that this association was independent of neuropathy effects ( $p$  less than 0.001). Several changes in individual manometric parameters related to neuropathy alone were appreciated only when the patients with psychiatric illness were excluded from the analysis. These data indicate that some of the esophageal neuromuscular dysfunction observed in diabetics is independent of neuropathy yet is strongly associated with psychiatric disorder. Such findings help to clarify the discrepant relationship of motility disturbances to neuropathy noted in prior reports. We conclude that consideration should be given to psychiatric illness as well as to neuropathy when interpreting manometric features suggestive of autonomic dysfunction in diabetic patients.

L22 ANSWER 27 OF 29 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 84128913 EMBASE

DOCUMENT NUMBER: 1984128913

TITLE: [Cardiovascular reflexes, vibration threshold and electroneurographic parameters of the peroneus nerve and sural nerve in Type I diabetes].  
 UNTERSUCHUNG DER KARDIOVASKULAREN REFLEXE, DER VIBRATIONSSCHWELLE UND DER ELEKTRONEUROGRAPHISCHEN PARAMETER DES N. PERONAEUS UND SURALIS BEI TYP-I-DIABETIKERN.

AUTHOR: Mayr N.; Wimberger D.; Mamoli B.; Vierhapper H.

CORPORATE SOURCE: Abteilung für Elektro-Neuro-Diagnostik der Neurologischen Universitätsklinik, A-1090 Wien, Austria

SOURCE: Wiener Klinische Wochenschrift, (1984) 96/10 (393-398).  
 CODEN: WKWOAO

COUNTRY: Austria

DOCUMENT TYPE: Journal

FILE SEGMENT: 008 Neurology and Neurosurgery  
 006 Internal Medicine  
 018 Cardiovascular Diseases and Cardiovascular Surgery  
 003 Endocrinology

LANGUAGE: German

SUMMARY LANGUAGE: English

AB In 26 type I diabetics ranging in age from 18 to 65 years with a duration of illness of between 1 and 34 years the following investigations were performed: 1. case history and questionnaire; 2. neurological examination; 3. determination of the vibration threshold; 4. electroneurography of the peroneal nerve and the sural nerve; 5. determination of the cardiovascular reflexes; 6. medical examination and additional findings; 7. ophthalmological investigation. 21 patients showed evidence of sensorimotor polyneuropathy (SM-PNP), the average age of this group (41 years) being 10 years higher than in the group without SM-PNP (31 years). The values of HbA1(c) were pathological in 17 of 21 cases with SM-PNP, and 2 of 5 cases without SM-PNP. Retinopathy was found rarely in either group. 13 patients showed evidence of autonomic neuropathy (ANP). The mean duration of illness (15.4 years) and the average age of the patients (36.5 years) in this group was distinctly higher than in the group without ANP (mean duration of illness: 8.9 years, mean age: 31.9 years). 12 patients with ANP and 7 patients without ANP had abnormally high HbA1(c) levels. Diabetic SM-PNP was most frequently (in 19 of 21 cases) diagnosed by electroneurographical investigation of the peroneal nerve. In the diagnosis of diabetic ANP the anamnesis (8 positive findings) and the determination of the heart rate variation during deep breathing (7 positive findings) are complementary. Among the 13 patients

with ANP, 12 also had SM-PNP, whereas among the 21 patients with SM-PNP only 12 showed evidence of concomitant ANP.

L22 ANSWER 28 OF 29 MEDLINE DUPLICATE 11  
ACCESSION NUMBER: 81139288 MEDLINE  
DOCUMENT NUMBER: 81139288 PubMed ID: 7202858  
TITLE: Comparison of clinical course and sequential electrophysiological tests in diabetics with symptomatic polyneuropathy and its implications for clinical trials.  
AUTHOR: Greene D A; Brown M J; Braunstein S N; Schwartz S S; Asbury A K; Winegrad A I  
CONTRACT NUMBER: MD1 RR00040 (NCRR)  
P30 AM 19525 (NIADDK)  
SOURCE: DIABETES, (1981 Feb) 30 (2) 139-47.  
Journal code: 0372763. ISSN: 0012-1797.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: (CLINICAL TRIAL)  
Journal; Article; (JOURNAL ARTICLE)  
(RANDOMIZED CONTROLLED TRIAL)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 198105  
ENTRY DATE: Entered STN: 19900316  
Last Updated on STN: 19980206  
Entered Medline: 19810526

AB The use of electrophysiological (EP) tests as the primary basis for determining outcome in clinical trials of therapy for symptomatic diabetic polyneuropathy, and the frequently short duration of such trials, is based on assumptions at variance with the pathology and natural history of this disorder and with the evidence that the commonly employed EP tests predominantly reflect the status of the large myelinated nerve fibers. The course of painful, distal symmetrical, primarily sensory polyneuropathy was studied in nine chronic diabetics, aged 21--59 yr, selected for the absence of other forms of diabetic neuropathy, other causes of **neuropathy**, and other significant **illness**. All were treated with modifications of diet, **insulin**, and a daily multivitamin tablet, and, on a randomized basis, also received either placebo or myo-inositol tablets. Initially, and after 2, 4, and 6 mo, a standardized questionnaire was used to assess symptoms, and a standardized neurological examination and battery of EP tests were performed. A minimum of 6 mo was found necessary to assess the clinical course of this syndrome. Clinical improvement occurred in both legs and arms in four patients, as judged by improvement both in symptoms and in the extent of deficits in pinprick and temperature perception; abnormalities in sensory modalities mediated by large myelinated fibers, however, were generally unaltered after 6 mo. A nonuniform distribution of abnormal EP tests of sensory components of the commonly studied nerves of the leg and arm was demonstrated in the study group at the outset, and clinical improvement was not accompanied by evidence of any consistent pattern of improvement in the initially abnormal EP tests. A significant fraction of chronic diabetics with painful, distal symmetrical, primarily sensory polyneuropathy selected by standard criteria appear to have potential for clinical improvement over 6 mo, but primarily in sensory modalities that make it inappropriate to use the common EP tests as the primary basis of judging outcome.

L22 ANSWER 29 OF 29 MEDLINE DUPLICATE 12  
ACCESSION NUMBER: 82022297 MEDLINE  
DOCUMENT NUMBER: 82022297 PubMed ID: 7283398  
TITLE: Seizures, hypoxic-ischemic brain injury, and intraventricular hemorrhage in the newborn.

AUTHOR: Hill A; Volpe J J  
SOURCE: ANNALS OF NEUROLOGY, (1981 Aug) 10 (2) 109-21.  
Journal code: 7707449. ISSN: 0364-5134.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 198111  
ENTRY DATE: Entered STN: 19900316  
Last Updated on STN: 20000303  
Entered Medline: 19811122

AB The review deals with neonatal seizures, perinatal hypoxic-ischemic brain injury, and neonatal intraventricular hemorrhage. neonatal seizures are the most prominent signals of the largest number of neonatal neurological disorders. The convulsive phenomena may be subtle. The predominant etiological process is hypoxic-ischemic encephalopathy. Prognosis is related primarily to the neurological disease responsible for the seizures. Treatment may be specific for the underlying disorder (e.g., **glucose** or calcium) or less specific (i.e., therapy with anticonvulsant drugs). Prompt control of the seizures is important to avoid brain injury secondary to the effects of the seizures on ventilation, perfusion, and brain metabolism. Hypoxic-ischemic encephalopathy in the newborn most often is a consequence of intrauterine asphyxia. Diagnosis depends primarily on recognition of the clinical syndrome but also on a variety of neurodiagnostic techniques, including radionuclide and CT brain scans. Prognosis is estimated best by a combination of clinical analysis and specialized neurodiagnostic studies. management is based principally on vigorous support, particularly of ventilation and perfusion, maintenance of adequate **glucose** influx, and control of seizures. Intraventricular hemorrhage is the most common type of neonatal intracranial hemorrhage. The **neuropathology** is characterized by bleeding from capillaries of the subependymal germinal matrix. Secondary rupture of the ependymal lining then causes intraventricular hemorrhage. Pathogenesis relates to the anatomy of the germinal matrix, the distribution and regulation of cerebral blood flow, and the structure and vulnerability of periventricular capillaries. Precise diagnosis requires a brain imaging procedure; portable, real-time ultrasound is the preferred approach for **critically** ill infants. Prognosis relates to the severity of the hemorrhage as well as any preceding hypoxic-ischemic insults and the subsequent occurrence of hydrocephalus. Choice of therapy for posthemorrhagic ventricular dilatation depends upon severity and rapidity of progression and ranges from close observation only to ventriculoperitoneal shunting.